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Effect of *Caesalpinia pulcherrima* (L.) Sw. seeds on serum glucose and other metabolic parameters of normal and alloxan - induced diabetic rats

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ABSTRACTS

Oral administration of the ethanol extract of *Caesalpinia pulcherrima* seeds (CP - 250 and 500 mg/kg) caused significant fall in blood glucose levels even at 2½ h after a single dose of treatment in normal fasted and glucose loaded Wistar albino rats. At 250 mg/kg dose level, CP completely prevented the elevation of blood glucose caused by oral glucose feeding. In alloxan diabetic rats, CP was able to lower the blood glucose level to around 132 mg / 100 ml from 10th day and thereafter. The biochemical findings were supported by histopathological studies of liver, kidney and pancreas of control and treated rats. CP was able to increase catalase levels of diabetic rats. Reduced levels of serum protein and elevated levels of Aspartate aminotransferase (AST), Alanine transaminase (ALT), alkaline phosphatase(ALP), cholesterol, triglycerides, creatinine and uric acid were almost normalised in CP treated diabetic rats. CP was also able to reduce *in vitro* lipid peroxidation in rat liver microsomes and inhibit 1- diphenyl – 2-picryl hydrazyl (DPPH) induced free radicals significantly.

Keywords: *Caesalpinia pulcherrima,* anti-diabetic, anti-oxidant, alloxan, lipid peroxidation, DPPH quenching,

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INTRODUCTION

Medicinal plants are Nature's gift to mankind and they form part of the rich heritage of India. Plant drugs are considered to be less toxic and free from side effects than synthetic ones.[1] Many Indian medicinal plants have been found to be useful in successfully managing diabetes and from some of them, active principles have been isolated.[2] It is well known that herbal plants like *Garlic* (*Allium sativum*), Tulsi (*Ocimum sanctum*), Neem (*Azadirachta indica*) and Bitter gourd (*Momordica charantia*) not only possess hypoglycaemic activity but some of them are hypotensive, hepatoprotective and also blood purifiers.[1] *Caesalpinia pulcherrima* (L.) Sw. belonging to the family Caesalpinaceae is known as 'Peacock Flower' or 'Red Bird of Paradise' in English and 'Rajamalli' in Malayalam. It is an exotic, hardy shrub or small tree, growing up to 5 m in height and cultivated in gardens throughout India. The seeds of *C pulcherrima* is used in traditional medicine of Kerala to treat diabetes. The roots are used as a remedy for lung and skin diseases. They are prescribed as a decoction for intermittent fevers and in the powdered form for infantile convulsions. The dried and powdered leaves are used to treat in erysipelas. The bark is higly astringent and widely used as an emmenogogue. The flowers are a remedy for intestinal worms. The anti - diabetic effects of *C pulcherima* have not scientifically validated yet. In the present study, the effect of *Caesalpinia pulcherrima* seeds on metabolic parameters of normal and alloxan – induced diabetic rats is reported.

MATERIALS AND METHODS

Plant material and preparation of the extract

The seeds of *Caesalpinia pulcherrima* were collected from Palode, Thiruvananthapuram District, Kerala. They were authenticated by the plant taxonomist of the Institute and a voucher specimen (TBGT 57024 dated 17/ 04 / 07) was deposited at the Institute's Herbarium. The seeds were shade dried and powdered. The powder (100 gm) was successively extracted with 1000 ml of ethanol overnight, at room temperature with constant stirring. The extract was filtered and the filtrate concentrated under reduced pressure to yield 750 mg of the crude extract (0.75 % with respect to the dried plant material). This crude extract was referred to as CP. It was reconstituted in 0.25% Tween-80, to desired concentrations and used for the experiments.

Animals

Wistar albino rats, males (250 - 300g) and Swiss albino mice, males (25 – 30 g), were obtained from the Institute's Animal House. They were housed under standard laboratory conditions (temperature

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24 - 28°C, relative humidity 60 – 70 % and 12 h dark and light cycles) and fed commercial rat feed (Lipton India Ltd., Mumbai, India) and boiled water, *ad libitum*. All experiments involving animals were carried out according to NIH guidelines, after getting the approval of the Institute's Animal Ethics Committee.

Effect of crude extract (CP) on glucose tolerance

Rats were divided into 4 groups, of six animals and fasted overnight. Group I, the vehicle control received 1ml of 0.25 % Tween - 80.Groups II and III were given po, the crude extract, CP (250 and 500 mg/kg). Glibenclamide (600 mg /kg) in 10%Tween - 80 was given to the positive control to group IV. Rats of all the groups were loaded with 60 % glucose (3 g /kg, po), 30 min. after CP administration. Blood samples were collected from the orbital plexus[4] just prior to drug administration and at 30 min, 90 and 150 min after glucose loading and glucose levels were determined spectroscopically.

Hypoglycaemic study in normal fasted rats with CP

Overnight fasted rats were divided into 4 groups of 6 rats each. Group I received 1ml of 0.25% Tween - 80 (control) and groups II and III received orally CP at 250 mg/kg and 500 mg/kg doses. Blood samples were collected from the orbital plexus at 60 and 120 min. after CP administration and glucose levels were measured spectroscopically. Glibenclamide (600 mg/kg) was administered to Group IV animals (positive control).

Effect of CP on alloxan induced diabetic rats

Rats were made diabetic by injecting alloxan (60 mg / kg)[5] through tail vein. 5 days later, blood samples were drawn and glucose levels were determined and the diabetic rats exhibiting glucose levels in the range of 400 – 450 mg /100 ml were selected for further studies.

Of the 5 experimental groups, Group I comprised of 6 normal non diabetic control animals and were given a single daily dose of 0.25 % Tween - 80 (1 ml, po), Alloxan induced diabetic rats were divided into 4 groups of 6 rats each (Groups II - V). Group II (alloxan diabetic control) received a single daily dose of 0.25% Tween - 80 (1ml, po), Groups III and IV were administered a single daily dose of CP (250 and 500 mg / kg, po respectively) and GroupV animals received po, 600 mg / kg of the standard drug, glibenclamide. The treatments were continued for 15 days. Blood samples were collected in the morning, 1 h after CP administration on day 1, 4, 7, 10 and 15.

Biochemical and histological studies

On day 15th, animals were sacrificed by mild ether anesthesia and blood was collected from all the five groups, for biochemical studies and liver, pancreas and kidney samples were collected for histological studies.

Biochemical parameters like Alanine transaminase (ALT), Aspartate aminotransferase (AST), serum alkaline phosphatase (ALP), cholesterol, total protein, creatinine, uric acid and triglycerides were assayed according to standard methods.[6-12] Seven µm thick paraffin sections of buffered formalin - fixed liver, pancreas and kidney samples were stained with haematoxylin - eosin for photomicroscopic observations of the organ histological architecture, of the control and treated rats.

Antilipid peroxidation studies

The antilipid peroxidant effect of CP was studied *in vitro*, following modified methods.[13,14] Briefly, 0.5 gm of the rat liver tissue was homogenized with 10 ml of 150 mM KCl-Tris-HCl buffer (pH 7.2). 0.25 ml of liver homogenate, Tris - HCl buffer (pH 7.2), 0.05 ml of 0.1 mM ascorbic acid (AA), 0.05 ml of 4 mM $FeCl_2$ and 0.05 ml of varying concentrations of CP extract. The mixture (in triplicate) was incubated at 37°C for 1 hr in capped tubes. Then 0.5 ml of 0.1 N HCl, 0.02 ml of 9.8% sodium dodecyl sulphate (SDS), 0.9 ml of distilled water and 2 ml of 0.6% thiobarbituric acid (TBA) was added to each tube and vigorously shaken. All the tubes were then placed in a boiling water bath at 100°C for 30 min. After cooling the flocculent precipitate was removed by adding 5 ml of n - butanol and they were centrifuged at 3000 rpm for 20 min. The absorbance of the supernatant was measured at 532 nm.

DPPH radical scavenging activity

DPPH radical scavenging activity was measured by the spectrophotometric method.[15] To an ethanol solution of DPPH (200 µ*M*), 0.05 ml of CP dissolved in ethanol were added at different concentrations (100 - 500 µg/ ml). An equal amount of ethanol was added to the control. After 20 min, the decrease in the absorbance of the test mixture (CP) (due to quenching of DPPH free radicals) was read at 517 nm and the percentage inhibition calculated by using the formula[16] given below.

Behavioural and toxic effects

four groups of 12 mice were administered p.o. 250, 500, 1000 and 1500 mg/kg of the CP extract. They were observed continuously for 1 h for any gross behavioral changes, symptoms of toxicity and mortality if any and intermittently for the next 6 h and then again, 24 h after dosing with CP extract.

Statistical analysis

Data were expressed as mean ± standard deviation of the mean (SD), and statistical comparisons were performed using Student's't' test.[17]

RESULTS

Effect of CP on blood glucose levels of normal fasted and glucose loaded rats

Oral administration of CP at 250 mg/kg showed significant fall in blood glucose level, 2 ½ hours after a single dose of treatment in normal fasted and glucose loaded rats. CP was effective at 250 mg/kg dose in depressing the value of blood glucose at 30 min. after glucose loading (Table 1).

Effect of CP on blood glucose levels of hypoglycaemic rats

CP showed significant hypoglycaemic activity at 250 mg/kg dose and decreased the blood glucose level significantly up to 120 min after administration (Table 2).

Effect of CP on blood glucose levels of alloxan - diabetic rats

The blood glucose levels of alloxan - diabetic rats was in the range of 298 – 412 mg/ 100ml. In the CP treated group, blood glucose level steadily decreased and it was around 132 mg/ 100 ml on the 10^{th} day. On the 15^{th} day, blood glucose level decreased to around 120 mg/100ml. (Table 3).

Effect of CP on other serum biochemical parameters of alloxan diabetic rats

A significant increase in the Alanine transaminase (ALT), Aspartate aminotransferase (AST) and alkaline phosphatase (ALP) activities were recorded in diabetic rats compared to normal rats, indicating an altered liver function in the diabetic condition. But a significant reduction in the values of ALT, AST and ALP were collectively indicates that the restorations of its activities were found in both CP treated groups compared to diabetic rats. ALT level in group which is treated with CP 500mg/kg was found to be 57.31 ± 6.09 which is closer to normal control 83.31 ± 6.12 than the animals treated with CP 250mg/kg their level was 52.32 ± 6.12. AST levels were found to be more or less same in all CP and Glibenclamide treated groups and but significantly reduced when compared with diabetic control group. The results of Group 3, treated with CP 250mg/kg showed that the ALP level was near normal as in the cases of normal control and Glibenclamide treated group as well. Serum levels of total cholesterol, triglyceride, creatinine and uric acid levels were found to be significantly increased with lowered total protein concentration in the diabetic group. Upon treated with two different dosage levels of CP 250mg/kg and 500mg/kg, the results showed that significant normalization of these elevated serum parameter levels with increased total protein concentration when compared with diabetic control. The results have clearly indicates that the restoration to near normal levels of total cholesterol (50.71±6.09), creatinine (0.59±0.10) and uric acid levels (0.67±0.12) were found in CP with dosage level of 250mg/kg treated group. Meanwhile, all other serum biochemical parameters were also found to be more or less at near normal invariably in all the drug treated alloxan induced diabetic rats. (Tables 4 and 5).

Effect of CP on hepatic catalase, glycogen and protein levels

Administration of CP at 250 and 500 mg/kg dose raised the activity of CAT in liver tissue of diabetic animals. There was a significant increase in the values of liver glycogen and liver protein in the CP and standard antidiabetic drug glibenclamide treated groups (Table 6).

Histopathology

Histopathological observations basically supported the serum parameter assays. Pancreatic sections of diabetic rats showed atrophy of β – cells and vascular degenerative changes in islets. CP and standard antidiabetic drug glibenclamide treated groups treated pancreas showed increase in the islets. Liver tissue of the diabetic rats showed distortion in the arrangement of cells around the central vein and fibrosis. CP treatment restored the cellular arrangement and reduced fibrosis. Kidney sections of diabetic animals showed wall thickening of nephrons and glomerulopathy. CP treatmet reversed these changes significantly (Figures 1 – 3, a - d).

Effect of CP on in vitro lipid peroxidation

CP showed very potent inhibition of $FeCl_2$ - AA stimulated rat liver lipid peroxidation *in vitro* at concentrations of 1-10 µg /ml. There was a significant increase of malondialdehyde (MDA) in $FeCl_2$-AA treated rat liver homogenate, compared to normal control without $FeCl_2$ - AA (Table 7).

Effect of CP on DPPH free radical scavenging activity

CP showed maximum inhibition (75.37 %) of DPPH free radical at 400 µg /ml. 25 µg /ml dose failed to evoke significant response and it was observed that the free radical was scavenged in a concentration dependent manner upto 400 µg /ml. (Table 8).

Table 1 **Effect of ethanol extract of *Caesalpinia pulcherrima* seeds (CP) on blood glucose levels of normal fasted and glucose loaded rats. [Values are mean ± SD of 6 animals]**

Groups	Blood glucose (mg/100 ml)			
	0 min	30 min	90 min	150 min
Normal control (0.25 % Tween – 80)	56.0 ± 5.0	141.0 ± 3.2	133.0 ± 4.2	115.0 ± 1.2
CP(250 mg/kg)	58.9 ± 1.4	82.3 ± 1.7**	86.3 ± 1.6**	81.4 ± 2.1**
CP(500 mg/kg)	53.6±1.2	108.3 ± 1.2**	109.3 ± 1.6**	106.3 ± 1.2**
Glibenclamide (600 mg/kg)	60.1±1.75	58.1 ± 1.3**	40.0 ± 1.2**	40.0 ± 1.3**

** Significance $P \leq 0.01$, compared to normal control group

Table 2 **Effect of ethanol extract of *Caesalpinia pulcherrima* (CP) on blood glucose levels of hypoglycaemic rats. [Values are mean ± SD of 6 animals]**

Groups	Blood glucose (mg/100 ml)		
	0 min	60 min	120 min
Normal control (0.25 % Tween – 80)	67.4 ± 1.8	63.2 ± 1.7	63.4 ± 1.3
CP(250 mg/kg)	66.3 ± 1.6	46.0 ± 0.8**	51.8 ± 0.9**
CP(500 mg/kg)	63.2 ± 1.6	49.3 ± 0.7	54.3 ± 1.0**
Glibenclamide (600mg/kg)	65.1 ± 1.3	58.1 ± 1.3**	60.0 ± 1.3**

** Significance $P \leq 0.01$, compared to normal control group

Table 3 **Effect of ethanol extract of *Caesalpinia pulcherrima* (CP) on blood glucose levels of alloxan - diabetic rats. [Values are mean ± SD of 6 animals]**

Groups	Blood glucose levels (mg / 100ml)				
	Day 1	Day 4	Day 7	Day 10	Day 15
Normal Control (0.25% Tween 80)	88.0 ± 4.2	89.3 ± 3.9	85.3 ± 4.0	88.0 ±5 .1	86.0 ± 4.0
Alloxan Control (60 mg / kg)	298.0 ± 6.3	372.1 ± 2.6	412.3 ± 1.5	365.2 ± 1.8	363.2 ± 1.9
CP (250 mg/kg)	284. ± 6.1	210.2 ± 5.1**	170.2 ± 3.4**	132.2 ± 1.6**	120.1 ± 4.3**
CP (500 mg/kg)	216.3 ± 4.3	220.2 ± 3.9**	182.8 ± 4.2**	138.1 ± 2.3**	126.3 ± 2.1**
Glibenclamide (600mg/kg)	286.1 ± 2.3**	207.1 ± 2.1**	166.4 ± 2.1**	128.2 ± 4.3**	115.3 ± 4.1**

** Significance $P \leq 0.01$, compared to alloxan control group

Table 4 **Effect of ethanol extract of *Caesalpinia pulcherrima* (CP) on other serum biochemical parameters of alloxan diabetic rats. [Values are mean ± SD of 6 animals]**

Groups	ALT (IU/L)	AST (IU/ L)	ALP (IU/L)
Normal Control (0.25% Tween 80)	83.31 ± 6.12	123.21 ± 6.23	207.31 ± 9.01
Alloxan Control (60 mg / kg)	142.23 ± 7.11	251.32 ± 6.09	299.32 ± 6.12
Alloxan + CP (250 mg/kg)	52.32 ± 6.12**	200.31 ± 8.02**	214.36 ± 7.23**
Alloxan + CP (500 mg/kg)	57.31 ± 6.09**	210.26 ± 3.12**	220.71 ± 6.21**
Alloxan + Glibenclamide (600mg/kg)	61.32 ± 7.11**	203.26 ± 9.12**	209.73 ± 8.17**

** Significance $P \leq 0.01$, compared to alloxan control group

Table 5 **Effect of ethanol extract of *Caesalpinia pulcherrima* seed extract (CP) on rat serum cholesterol, total protein, triglycerides, creatinine and uric acid after alloxan administration. [Values are mean ± SD of 6 animals]**

Groups	Cholesterol (mg / dl)	Total Protein (mg / dl)	Triglycerides (mg / 100 ml)	Creatinine (mg / 100ml)	Uric acid (mg / 100ml)
Normal Control (0.25% Tween 80)	50.31±9.27	407.31±8.29	86.83±5.5	0.52±0.1	0.68±0.016
Alloxan control (60 mg / kg)	87.33±8.19	373.36±8.12	200.83±11.1	1.35±0.1	0.556±0.055
Alloxan + CP (250 mg/kg)	50.71±6.09**	398.31±6.21**	110.31±6.26**	0.59±0.10**	0.67±0.12**
Alloxan + CP (500 mg/kg)	53.32±7.13**	395.32±6.11**	117.61±3.15**	0.65±0.12**	0.63±0.017**
Alloxan + Glibenclamide (600mg/kg)	45.71±8.11**	390.33±9.09**	108.00±6.1**	0.58±0.1**	0.63±0.006**

** Significance $P \leq 0.01$, compared to alloxan control.

Table 6 **Effect of ethanol extract of *Caesalpinia pulcherrima* seed extract (CP) on hepatic catalase, glycogen and protein levels. [Values are mean ± SD of 6 animals]**

Groups	Catalase (mg / g)	Glycogen (mg / g)	Protein (mg / g)
Normal Control (0.25%Tween- 80)	3.00 ± 0.10	50.00 ± 2.00	16.00 ± 1.20
Alloxan control (60 mg / kg)	0.88 ± 0.01	26.00 ± 1.50	8.60 ± 2.60
Alloxan + CP (250 mg/kg)	2.75 ± 0.02**	38.00 ± 3.60**	14.20 ± 1.30**
Alloxan + CP (500 mg/kg)	2.52 ± 0.004**	32.00 ± 3.60**	12.50 ± 1.20**
Alloxan + Glibenclamide (600mg/kg)	2.45 ± 0.05**	37.00 ± 4.00**	12.00 ± 1.20**

** Significance $P \leq 0.01$, compared to alloxan control

Table 7 **Effect of ethanol extract of *Caesalpinia pulcherrima* seed extract (CP) on $FeCl_2$ - ascorbic acid (AA) - induced lipid peroxidation in rat liver homogenate *in vitro*. [Values are mean ± SD of 3 experiments]**

Groups	CP Concentration (mg / ml)	MDA (n mole / g wet liver)	MDA Inhibition (%)
Normal Control	-	1.34 ± 0.60	-
$FeCl_2$ - AA control	-	2.69 ± 0.02	-
$FeCl_2$ – AA + CP	1.00	1.89 ± 0.02	49.60**
$FeCl_2$ – AA + CP	5.00	1.63 ± 0.01	56.53**
$FeCl_2$ - AA + CP	10.00	1.96 ± 0.02	47.73**

** Significance $P \leq 0.01$,compared to $FeCl_2$ - AA Control

Table 8 **Effect of ethanol extract of *Caesalpinia pulcherrima* seed extract (CP) on DPPH radical scavenging activity [Values are mean of 3 experiments]**

Conc (µg/ml)	DPPH (% Inhibition)
25	42.75
50	63.75
100	62.75
200	70.00
400	75.37
800	73.77

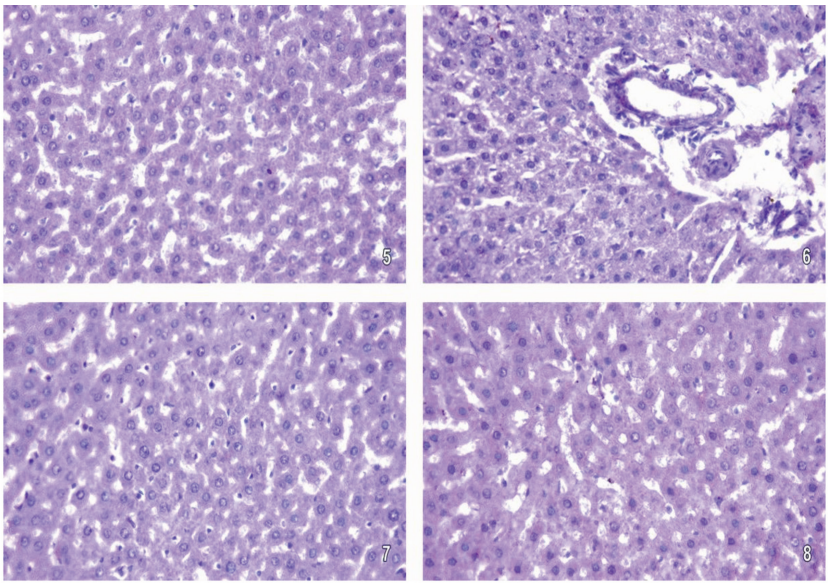

Figure 1 Histological evidence of the protective effect of ethanol extract of seeds of Caesalpinia pulcherrima (CP) /glibenclamide on liver of alloxan induced diabetes in Wistar rats. (a). Normal control showing hepatocytes with well brought out nuclei and cytoplasm (x 300). (b). Liver of diabetic rat showing degenerated parenchymatous cells with severe necrosis and dilation of sinusoids (x 300). (c). Liver of diabetic rat after treatment with CP, showing hepatocytes with nearly normal appearance and minimal necrosis(x 300). (d). Liver of diabetic rat after treatment with glibenclamide showing normal hepatocytes (x 300)

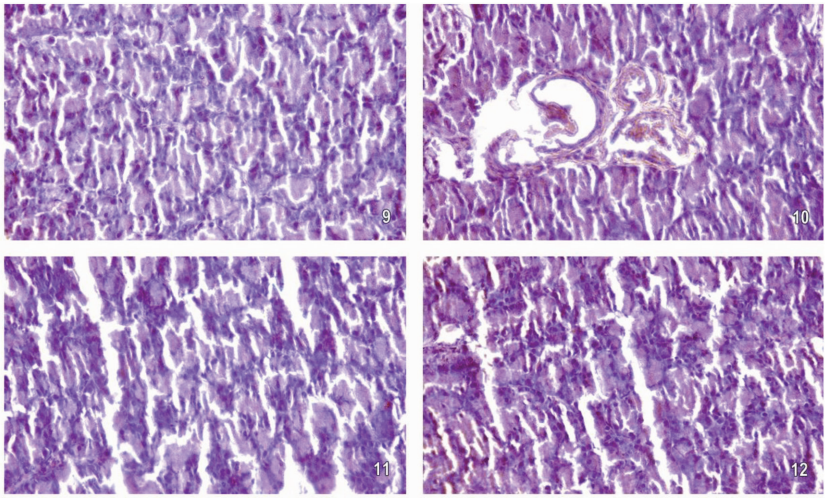

Figure 2 Histological evidence of the protective effect of ethanol extract of seeds of Caesalpinia pulcherrima (CP) / glibenclamide on pancreas of alloxan induced diabetes in Wistar rats. (a). Normal control showing abundant patches of β - cells (x 300). (b). Pancreas of diabetic rat showing minimal pancreatic β - cells (x 300). (c). Pancreas of diabetic rat after treatment with CP seed extract showing pancreatic β - cells almost similar to that of control (x 300). (d). Pancreas of diabetic rat after treatment with glibenclamide, showing pancreatic β - cells similar to that of the control (x 300)

Toxicity studies

In the acute toxicity studies, CP was found to be nontoxic up to the highest dose used (1500 mg /kg), The LD_{50} of CP was therefore above 1500 mg /kg in mice (data not shown).

DISCUSSION

Diabetes Mellitus (DM) is a major public problem world wide.[18] Many oral hypoglycemic agents are not very effective in lowering glucose in diabetic patients.[19] Therefore, the search for effective drugs for the treatment of diabetes mellitus continues. Many herbs have been shown to have hypoglycemic action in animals and humans.[20,21] There are several reports on the antidiabetic properties of *Terminalia catappa, Momordica charantia*[22] *elumbo nucifera*[23] etc. The present study reports for the first time the anti - hyperglycemic and anti - diabetic effects of *Caesalpinia pulcherrima* in rats. CP exhibited anti - hyperglycemic effects in normal fasted and alloxan induced diabetic rats. The effect was comparable to glibenclamide, the positive control used in this study. Alloxan induces diabetes by destroying the b cells and the destruction is almost complete and it impairs renal function. In the present study, CP significantly reduced the blood glucose levels of alloxan diabetic rats indicating the mechanism of action may be due to potentiation of insulin release from the surviving pancreatic cells, but due possibly to increased peripheral glucose utilization and inhibition of proximal tubular re absorption of glucose in the kidney.[24]

CP was also found to be an effective agent against other metabolic alterations induced as a consequence of diabetes. Insulin deficiency leads to various metabolic alterations in the animals like increased blood, cholesterol, triglycerides, AST, ALT, ALP and decreased levels of serum proteins.[25] In the present study, the levels of all the above parameters were almost normalized by CP treatment this is due to an improvement in carbohydrate, fat and protein metabolism. The restoration of AST and ALP after treatment with CP also indicates a revival in insulin secretion. CP was able to increase the levels of catalase of diabetic rats, thereby helping to detoxify the deleterious free radicals. It may prevent the formation of free radicals or it may scavenge the reactive oxygen metabolites through its antioxidant phytocompounds. In chronically diabetic animals, with high glucose levels, a decrease in liver glycogen could be attributed to the lesser availability of the enzyme glycogen synthase which in turn has been reported to be responsible

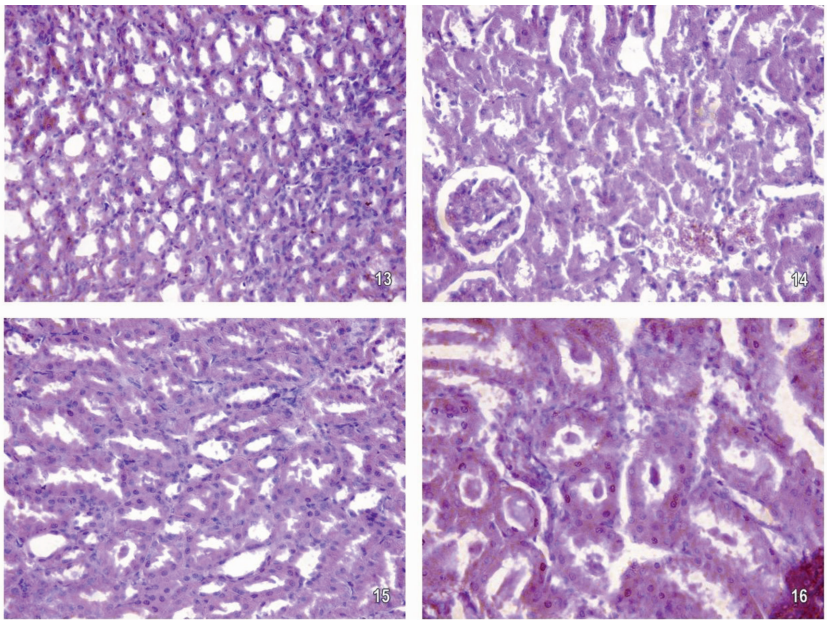

Figure 3 Histological evidence of the protective effect of ethanol extract of seeds of Caesalpinia pulcherrima (CP) / glibenclamide on kidney of alloxan induced diabetes in Wistar rats.(a).Normal control kidney showing normal Bowman's capsule and renal tubules (x 300). (b). Kidney of diabetic rat showing degenerative changes, expanded glomerulus and thickening of the walls of renal tubules (x 300). (c). Kidney of diabetic rat after treatment with CP showing glomerulus with reduction in thickening of walls of renal tubules (x 300). (d). Kidney of diabetic rat after treatment with glibenclamide showing normalcy of Bowman's capsule (x 300)

Fig. 1 *Caesalpinia pulcherrima* l. Sw.; Fig. 2 Inflorescence; Fig. 3 Flower; Fig. 4 Fruits and seeds.

for the incorporation of glucose moieties into the preexisting glycogen chain. In the present study, the normalization of depressed glycogen level by CP may be due to the regulation of glucose metabolism as reported by Dhawan et al[26] with the herbal anti - diabetic formulation, D - 400.

Histopathological studies of pancreas, liver and kidney of control and CP treated diabetic rats, supported the results obtained from biochemical analysis CP treatment protected the pancreas which showed increased number of islets compared to alloxan diabetic control. Similarly the histopathological changes in the liver and kidneys were more or less normalized by CP treatment.

CONCLUSION

From the results, it is clear concluded that CP contained active phytocompounds capable of stimulating the surviving b cells of the pancreas damaged by alloxan, to release more insulin and perhaps the phytocompounds of CP were of an anti oxidant nature. Free radicals produced by alloxan are known to cause pancreatic damage. Phytochemical studies of *C pulcherrima* reported the presence of tannins and flavonoids[27] which are known to have antidiabetic properties.[28] CP also showed anti lipid peroxidation and free radical scavenging effects. Thus it acts as an antioxidant and protects the liver, kidneys and pancreas from free radical damage. Thus CP appears to be an attractive material for further studies, leading to possible drug development for diabetes.

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CONFLICT OF INTEREST

Authors do not have conflict of interest.

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